

LETTERS TO THE EDITORS

Regarding "Popliteal entrapment: more common than previously recognized"

To the Editors:

Drs Levien and Veller are to be commended for their excellent description of popliteal artery entrapment syndrome.¹ Their lucid description of the embryologic explanation significantly aids diagnosis and management of these anatomic abnormalities.

However, we remain unconvinced that all their patients suffered from this condition. They describe 30 limbs that were involved in a "functional" popliteal artery entrapment without evidence for an obvious developmental anatomic abnormality. In at least one of these patients unusual physical activity, most notably competitive bicycle riding, was alluded to as causing the symptoms (which abated with cessation of this activity). They state that only "moderate functional" popliteal artery entrapment syndrome was noted on angiography performed during plantar flexion.

We have had experience with another cyclist who, despite showing evidence of similar angiographic popliteal findings, ultimately developed an iliac artery thrombosis. In the absence of any other obvious etiology we concluded that this might have resulted from iliac artery trauma from extreme activity in the highly flexed position adopted by competitive cyclists. The patient, a 40-year-old completely healthy male, was a triathlete. He originally presented with claudication after 30 minutes of intense activity. Physiologic arterial studies with plantar flexion were normal, but the ankle/brachial index fell to 0.78 only after he ran for 30 minutes. Arterial duplex scan performed concurrently failed to demonstrate any changes in the popliteal artery. An angiogram was performed and demonstrated mild compression of a normally positioned popliteal artery with plantar flexion. Three years later he presented with a sudden onset of severe one-block claudication. Angiography now demonstrated a complete localized external iliac artery occlusion. Lytic therapy was initiated. The native arterial system was successfully opened, demonstrating a smooth, tapered, short, segmental external iliac stenosis, which was successfully dilated and stented. The artery has remained patent for 2 years. However, he again develops claudication with extreme exercise despite ultrasound-confirmed patency of the stent. It is hypothesized that the rigid stent prevents iliac artery dilation that may occur during extreme activity.

Iliac artery compression syndrome associated with extreme exercise, especially cycling, has been reported previously.^{2,3} Accordingly, we question whether such an iliac etiology may have been present in the author's series, thus contributing falsely to the suggested prevalence of popliteal artery entrapment syndrome. We also raise the possibility that other etiologies of lower extremity claudication in young adults, such as activity-related compartment syndromes, may also be misdiagnosed as "function-

al" popliteal artery entrapment syndrome.

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REFERENCES

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Reply

The comments made in the letter by Dr Russell Samson and Dr David Showalter are much appreciated.

All patients in our series presented with severe claudication that limited their sporting and, occasionally, their daily activities. As part of the initial diagnostic workup, each patient was evaluated using noninvasive studies that included tests intended to exclude hemodynamically significant aortoiliac inflow stenosis. Both limbs of all patients were evaluated by contrast arteriography, and in all cases the aortoiliac segment was carefully studied to exclude inflow disease before treating the patient for popliteal artery entrapment. In only one patient was a lesion of the aortoiliac segment detected, and in this female the pathology was stenosis due to fibromuscular dysplasia, which was subjected to successful balloon angioplasty. She has remained asymptomatic and with normal inflow on noninvasive tests after 5 years' follow-up.

We are very aware of the existence of the iliac artery compression syndrome that has been described in competitive cyclists. We have actively pursued such a diagnosis in our series, as we believe that the pathological changes in the arterial wall of such cases are similar to those degenerative changes that we have described in the popliteal artery with entrapment and in the subclavian artery with thoracic outlet syndrome.^{1,2}

The majority of our patients in whom a diagnosis of functional popliteal artery entrapment was made were referred after full evaluation at sports medicine clinics where compartmental syndromes and other more common causes of leg pain in the athlete had been excluded. In addition, virtually all patients diagnosed with symptomatic functional popliteal artery entrapment and treated with myotomy of the medial head of the gastrocnemius muscle have returned to full sporting activities after the surgery.

In conclusion, we have carefully excluded the iliac artery compression syndrome in our series of symptomatic